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Are Eating Disorders: Anorexia Nervosa, Bulimia And Binge Eating Genetically Caused?

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ARE EATING DISORDERS: ANOREXIA NERVOSA, BULIMIA AND BINGE
EATING GENETICALLY CAUSED?

BY

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Abstract

The reasons behind women dieting to the point of death were examined. Is it all in the name of the fashion industry –just a desire to emulate TV images –or is there is deeper, more genetic reason behind disordered eating? This report uncovers astounding new research that traces the roots of bulimia, binge eating, and anorexia nervosa to specific genes, responsible for the manufacture of Serotonin (5-HTP) in the brain. Some revolutionary medical researchers describe the brain chemical as “The master molecule” (Murray, 1998)

Shortly after the conclusion of this report, the author found research that states’ “genetic variation in [the] CART [gene] may contribute to human obesity” (Giles et al. [online] 2000)

Acknowledgements

This Thésis project proved a tremendous challenge for me; yet, were it not for the many individuals who helped guide, encourage and sustain me throughout the process of the report, my own efforts would have been in vain. Pastor at St. Joseph Parish in Carteret, Fr. Edward Puleo was my spiritual advisor, and brother on numerous occasions; Monsignor Donald Endebrock, Senior Priest at St. Joe's, was also always within arms reach to help make this journey more possible. In fact Fr. Endebrock became so available to me that on one occasion, I simply picked up my phone, and before I could even dial his number, there he was at the other end. At the time I thought I detected an initial air of suspicion in his voice as I spoke, seemingly from out of nowhere.

Another individual of the cloth, our own President Monsignor Robert Sheeran, unwittingly, provided me with the feeling of inclusion I missed (because my campus visits were generally in the evenings), when he sent a general letter to students during the Boland Hall crisis, earlier this year. I seized the opportunity to return written comments to Monsignor Sheeran and was surprised and delighted when he responded with a personal note. I certainly wish to apologize if after his second response to me I felt that I could send him an email or two that may have distracted him from his own, important deadlines.

Throughout this MACPC degree process, my professors and advisors were helpful and willing to share their professional experiences. I feel truly grateful for their openness. To Michael McGraw, Ph.D., my Thesis advisor and teacher, as well as Patricia Kuchon, Ph.D., my academic advisor and mentor rolled into one, I must say a special thank you for their dedication to keeping me focused by acknowledging my progress, from time-to-time.

I am grateful, also, to Russell, my only child who, despite his preoccupation with the teenage years, nonetheless found the time to encourage me and acknowledge my struggle; and to Nelu Romanitan, my constant companion and friend, who utilized his many years of experience as an Officer in the Romanian military, to keep me focused on the desired target.

Last, but not least, I would like to thank my brothers, Dennis and Keith, who both traveled this road ahead of me, and were able to give me guidance from time to time; my sister, Marcelle, who gave me invaluable assistance during the course of the preparation of this report; and of course, my mother, who with her intercessory prayers and willing advice, seemed ever-present during this difficult process. Indeed, every individual whose life, by the grace of God, at the precise moment that I needed to learn something more about myself or about the others with whom I am fortunate enough to share this society. Thank you all!

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Chapter I

INTRODUCTION

This chapter presents an overview of the research question: Are eating disorders such as anorexia nervosa, bulimia and binge eating more the result of genetic factors than social and psychological circumstances? The background section discusses the traditional belief that eating disorders are primarily the result of body image disturbance, which causes females to believe that their bodies are ugly if they do not look like the slim bodies displayed in magazines and on television. "These women are making the diet industries richer every year" according to the Administrative Director of the American Anorexia and Bulimia Association, Dr. Claire Mysko (Bennett, 2000, on-line). This section also discusses new directions in research that suggest individuals with eating disorders are, first and foremost, victims of a genetic disturbance that predisposes them to become problem eaters. Authors of genetic research believe that social circumstances are merely secondary. "Eating disorders have not . . . been viewed as heritable illnesses [but] recent . . . twin studies [support] the potential role of genetic transmission", says Dr Walter Kaye, one of the leaders in the genetic research theory. (Kaye, Lilienfeld, Berrettini, Strober, Devlin, Klump, Goldman, Bulik, Halmi, Fichter, Kaplan, Woodside, Treasure, Plotnicov, Pollice, Rao, Mcconaha (2000, on-line).

Background of the Problem

"More than 5 million Americans suffer from eating disorders, and thousands of them die every year" (as cited in Bennet, 2000, on-line). For many decades, medical scientists believed that the drive to be thin was the major cause of girls and women starving themselves—to death, in some cases. Consequently, the burning question in the minds of treatment specialists has been: What pushes women to the point of becoming anorexic or bulimic?

Until about 2 years ago, the search for answers to the cause of eating disorders centered primarily among young Caucasian girls and women in the entertainment industry. "Mention Anorexia or bulimia and most people think of desperate teenage girls, models smoking to stay thin, or ballerinas eating tissues to stop the hunger pangs. But today, a disproportionate number of males in London are being treated for eating disorders are gay." ("Eating disorders are not just a woman's problem", 2000, on-line).

To further complicate the problem, the question of baby bulimics recently appeared in the news and strengthened the growing feeling among some medical professionals that eating disorders might have more to do with biology and genes than was previously believed:

"Some children will bite, scream, kick and vomit to avoid eating. They are neither picky eaters nor victims of body image disturbance. They are suffering from feeding disorders as serious as anorexia nervosa," says a trained school psychologist at Tulane University, Dr. Cathleen Piazza, (as cited in Raphael, 2000, on-line). As a result of this growing evidence that there is much more than social circumstances involved in the onset of serious cases of eating disorders, some noted psychologists and

psychiatrists have redirected their focus to “chemicals found in the brain that are important for the control of our behavior”: (University of Pittsburgh School of Medicine, 2000, on-line).

According to a statement from COPE (2000):

A great deal of research has been conducted on chemicals found in the brain [that] are important for the control of our behavior. Serotonin is one such chemical, or neurotransmitter, that has been linked to the control of hunger, as well as the control of depression, anxiety, and obsessive and compulsive behaviors [and] we are looking to see how serotonin may be related to eating disorders (on-line).

In addition, Dr. Cynthia Bulik, (as cited in Joseph, 2000) an eating disorders expert at Virginia Commonwealth University, feels that “genetic and brain chemistry studies of bulimics could help determine at an early stage, those individuals who are predisposed to eating disorders so they could get treatment before they have a serious problem” (on-line). Bulik also believes that within 5 years, current ideas of what a healthy woman looks like will change.

But this trend of thought is not yet acceptable to all researchers. For example, Dr. Michael Strober, Director of the eating disorders program at UCLA’s Neuropsychiatric Institute, warns that all this talk of genetic predisposition will have no immediate impact on these diseases. The genetic theory “adds weight to the idea that there are hereditary influences that play a role, but we don’t know what genes they are,

or what [is the] combination of genes and environment," says Strober, (as cited in Chamberlain, 1998, on-line), in an article entitled "Bulimia's Genetic Trail".

Purpose of the Study

In the opinion of this author, new studies are needed because of the possibility that eating disorders may be a symptom of a genetic illness, rather than the result of "tremendous pressure on people, particularly women . . . to strive for perfection in appearance and other areas, no matter how mythical that ideal really is" (Bennett, 2000, on-line).

Precise findings about the origin of eating disorders would mean the development of more effective treatment methods, thereby preventing thousands of needless deaths each year.

The author hopes that this review of existing literature will show it is possible that the major determinant in long-term cases of eating disorders is a variant gene, rather than "the byproduct of a society that neurotically shuns fat people, making those of normal weight strive for super-model thinness" (Joseph, 2000, on-line).

Limitations of the Study

This review will be limited to individuals who have already accepted the diagnosis of eating disorders, since data is already available on the development and treatment of their illness.

Other limitations may exist because, according to Nicky Bryant, Chief Executive of the Eating Disorders Association ("Gene theory on eating disorders", 2000, on-line).

"There is the risk that people with eating disorders will read about this [genetic research theory] and feel that they are deficient and defective in some way," (on-line), causing them to withhold important information during testing.

More limitations may concern the ethical controversy over the rights to findings in genetic research. "The question of who's doing what, how much and with whom, is a loaded one," says a Health News article: How much should genetic research be shared? (Abel, 2000, on-line) The President of the United States and British Prime Minister have asked "private gene research companies to deposit their gene sequence data in a public domain called GENBANK, which would be accessible to the global scientific community" (on-line). This project is known as the Human Genome Project or HGP.

In the author's opinion, this means researchers wishing to follow the genetic theory may anticipate constraints of this nature, and choose areas of research, that are less likely to be controversial than human genetics. Thus the world may be denied vital answers to the plague of eating disorders.

Data Collection

In order to begin work on this study, the author launched an internet search under the keyword Health Issues, then narrowed to Women's' Health Issues because of traditional belief that these diseases affect only women. This course of action proved fruitful to a certain extent. Statistics about age and other social circumstances were easily available. However, it was not until the author redirected the search to genetic illnesses, that data about familial tendencies and heredity became evident.

One of the names that appeared in almost all of the genetic studies done on eating disorders was that of Dr. Walter Kaye, a researcher at the Medical School of the University of Pennsylvania. A call to the Medical School revealed that although Dr. Kaye was indeed involved in psychiatric studies to show genetic tendencies in eating disorders he was too busy to be available to the author, because he travels extensively. Instead, another researcher in the Department of Psychiatry suggested that Medline, a medical data bank on research studies, would provide valuable information about Kaye's studies on this subject.

The Medline search did provide current information, but one immediate set back was that the website required registration by medical professionals, and a fee for all documents after the initial printout. This roadblock was removed when the author discovered an alternate but similar website, Pub Med, managed by The National Library of Medicine, that permitted students doing research to register and access most of the available material. At each visit, the author was allowed to download and print one document. For the rest, the old method of handwriting, worked just fine.

I subsequently registered at MEDSCAPE and received "warm regards" from Joseph Hatfield at Member Communications. The e-mail also provided an 800 number and an invitation to call for further assistance.

Additional information was obtained from the research section of the Elizabeth Public Library in New Jersey, which is a repository for Government and Federal data, and The Seton Hall Walsh Library in South Orange, New Jersey, which provides copies of numerous theses written by our alumni.

Documents, such as: Essential Guide to Depression (American Medical Ass. 1998) Eating Disorders: The Facts (Abraham, & Llewellyn-Jones, 1997) provided much-needed answers.

For organization, style and adherence to APA guidelines, I consulted a very professional-looking thesis by Lawrence Kostigan, a former graduate student of Dr. McGraw.

Research Question

Are eating disorders caused by genetic factors? This study explores that question by looking at current research on the role of social circumstances, as well as genetics, in eating disorders.

Subsidiary Questions

To better understand the part that genes play in eating disorders the author reviews studies and other literature that address the following questions:

1. Who are the individuals most likely to fall victim to an eating disorder?
2. What part does body image disturbance play in the onset of eating disorders?
3. What are some effective approaches to treatment of these illnesses?

Definition of Terms

1. Additive: a substance added in small amounts to something else to improve, strengthen, or otherwise alter it.

2. Anorexia: an eating disorder [that] involves people who intentionally starve themselves when they are already underweight
3. Bulimia: an eating disorder in which people consume large amounts of food during 'binge' episodes in which they feel out of control.
4. Bivariate: a medical diagnosis that involves two diseases.
5. Co-morbidity: the presence of co-existing or additional diseases with reference to an initial diagnosis, or with reference to the condition that is the subject of the study.
6. Etiology: a branch of medical science dealing with the causes and origin of diseases.
7. Extant: still in existence, not destroyed, lost or extinct.
8. Feeding disorder: a problem in children who have the inability or lack of desire to eat.
9. Gene: a hereditary unit that occupies a specific location on a chromosome and determines a particular characteristic in an organism.
10. Gene therapy: the treatment of certain disorders, especially those caused by genetic anomalies or deficiencies, by introducing specific engineered genes into a patient's cells.
11. Genetic marker: a known DNA sequence associated with a particular gene or trait that is used to indicate the presence of that gene or trait.
12. Heritable: of being passed from one generation to the next.
13. Heritability: the quality of being heritable, a measure of the extent to which a gene influences a disease.

14. MRI: Magnetic Record Imaging
15. Obsessive: excessive in degree or nature.
16. Pathogenesis: development of morbid conditions or of disease
17. Pathophysiology: the physiology of disordered function.
18. Proband: the original person presenting a mental or physical disorder who serves as the basis for a hereditary or genetic study.
19. Serotonin: an organic compound found especially in the brain, blood serum and gastric mucous membranes and active in transmission of nerve impulses. 5-hydroxytryptamine
20. Variant: something that differs in form only slightly, from something else.
21. 5HT2A: hydroxytryptamine, a substance that constricts blood vessels and transmits nerve impulses. A by-product of serotonin, produced when the body attempts to self-regulate large quantities of serotonin lodged in the system.

Summary

Although individuals diagnosed with eating disorders suffer untold pain and anguish, it is also a fact that entire families bear the negative consequences of these diseases. Hence, it is essential that correct diagnosis be made of the exact causes of the problem so that sufferers and their families could obtain speedy and lasting relief.

Chapter II

REVIEW OF GENETIC RESEARCH

Introduction

This chapter discusses the relationship between genes and inheritance; and the rationale behind the use of twin studies and to learn more about how biogenetic inheritance could trigger fatal eating disorders. Next, the chapter discusses a review of twin studies and their limitations, if any (Bulik, Sullivan, Wade & Kendler, 1998). The author then quotes current genetic studies that claim bio-chemical predisposition is, indeed, a valid theory which will eventually lead to answers and cures for these diseases.

The Role of Genes

According to the American Medical Association's Essential Guide to Depression (1998), a gene determines which features individuals inherit from their parents. Eye color, gender, and all the features which make an individual unique, are determined by genes for those traits. Each body cell has between 50,000 and 100,000 genes that are made of a substance called deoxyribonucleic acid (DNA). Genes reside in the control center of the cell, or the "nucleus," on fine structures called chromosomes. All cells, except the ones that determine gender, have 46 chromosomes and each gene usually occupies a particular place on a certain chromosome. Each individual has his genes arranged in a completely individual way. Identical twins, however, possess each other's exact genetic makeup, and this is the only instance in which scientists can study

scenarios in which one twin develops an illness from an eating disorder while the other twin, with identical genes, does not.

To discuss the twin idea behind eating disorder research a little further. Identical twins have 100 percent of their genes alike while fraternal twins have only half their genes in common. Consequently, if bulimia in both twins is more common among identical pairs than fraternal pairs, then one can theorize that the reason behind the onset of bulimia is genetic. This belief in the validity of studies done on twins to determine genetic contribution to eating disorders is rapidly gaining attention.

A review of one such twin study done earlier this year through the Virginia Institute for Psychology (Bulik, Sullivan, Wade & Kendler, 1998, on-line), says twin studies were used to show the presence of genetic factors in many medical disorders and behavioral traits including eating disorders. However, the review also specifies, that "although twin studies are powerful tools, their methodology can be arcane and their implications easily misinterpreted" (on-line).

This review (Bulik et al, 1998, on-line) was unable to draw firm conclusions about the exact roles played by genes and environment in anorexia nervosa; but the review did confirm that bulimia nervosa has familial tendencies and may be aggravated by certain 'unique' environmental factors. The extent of the contribution of shared environment is not known, but according to the review, in the studies with the greatest statistical power, environment seems to be less important than a genetic history of the disease.

Although many individuals have a genetic sequence that does not predispose them to some serious illness, some people inherit from their parents, one or

more abnormal genes. These abnormal genes may lead to health problems. For example, people with hemophilia are born with an abnormal gene that prevents their bodies from producing blood clots when they bleed. Knowledge about how genes help in various bodily functions helps guide scientists during twin and other gene-based studies designed to determine whether or not genetics are a factor in eating disorders (American Medical Association's Essential Guide to Depression (1988).

This literature review does not dismiss entirely the role of environment in the onset of eating disorders, but there are case studies that show individuals for whom social factors are the predominant determinant in these diseases, do not go on to experience complete and lasting cures. In fact Tobin Levy, a bulimic individual, (as cited in Cohen & Levy, 1998) was left with an incomplete treatment of her disease, and the constant fear that someday the illness may control her life. The mother-daughter diary kept by Tobin and her mother shows that after years of therapy, the young girl was left with the lingering hope that someday she would be able to control her bingeing and purging and be able to live a normal, healthy, life. However, the results at the end of her treatment were at best inconclusive, because the approach that targeted low self-esteem and the misinterpretation of glamorous TV images did not prove successful.

Below, Jennifer Joseph, an ABC reporter, gives a summary of the Levy story:

Tobin Levy began her first diet the summer after sixth grade, when all of her friends' bodies were beginning to change. Like other girls her age, she desperately wanted to shed the baby fat from her 5 ½ -foot frame. But that

desperation led her from extreme dieting to a daily cycle of bingeing and purging. And one year later, she was vomiting as often as 30 times a day.

Levy is now 23 years old, but she still binges and purges once in a while. "I'll have a great three months where I don't do it, and then, well, it's hard" (Joseph, 1998, on-line).

Levy's story is a familiar one to the five million women and men in America who suffer from eating disorders. For decades, we've believed that eating disorders were the byproduct of a society that neurotically shuns fat people, making those of normal weight work for super-model thinness. But as many as 70 percent of all American women struggle daily with their weight and body image, while a relative minority end up with life-threatening eating disorders. What pushes women like Levy to the point of becoming anorexic or bulimic? Researchers now believe they've found one answer (Joseph, 1998, on-line).

The Role of Serotonin

In order to help prove the validity of the genetic theory, researchers at Maudsley Hospital are examining the role of the brain chemical, serotonin, ("Genetic clues to eating disorders", 1999, on-line).

"We were involved in a genetic study to look through the whole human genome to try and find genes for anorexia. The one we picked first was the 5HT2A receptor, [which] is involved in regulating feeding," says Dr. Collier, one of the Maudsley researchers

"Genetic clues," 1999, on-line). Dr. Collier and his colleagues already know that drugs that block serotonin cause individuals to gain weight.

Dr. Collier's study found that variations in the gene for serotonin receptors are twice as likely to appear in anorexic patients than in women without eating disorders. As a result of this study, Dr. Janet Treasure, Director of the Eating Disorders Unit at Maudsley, theorizes that this disabled gene for serotonin is the one responsible for suppressing the appetite of anorexic sufferers.

"In anorexia nervosa, the drive to eat can be inhibited, but we know that normal people who are starved . . . will kill each other and do all sorts of morally repugnant things . . . that you wouldn't normally touch" (Genetic clues," 1999, on-line).

Since this does not occur in patients with anorexia nervosa, "there's some aspect of the appetite system that isn't working", says Treasure, (as cited in "Genetic clues," 1999, on-line).

Earlier studies did find "abnormal levels of serotonin" in the brains of bulimic individuals but researchers were unsure whether the high levels of this chemical caused the disease or existed as a result of the eating disorder (Joseph, 1998, on-line).

Then Dr. Walter Kaye, (as cited in Joseph, 1998), a Psychiatry professor at the Pittsburgh Medical School, found that "bulimic women have altered brain chemistry, possibly from birth, which puts them at a higher risk for eating disorders --even long after they've recovered from bulimia" (on-line).

Dr. Kaye (as cited in Joseph, 1998) compared 31 healthy women with 30 women who were cured for 1 year. The study, originally published in the October 1999 issue of

the Archives of General Psychiatry, found that the bulimia survivors had twice the levels of a by product of serotonin (5-HIAA) than the normal population.

Previous studies found that bulimics' brains contained abnormal levels of serotonin, a chemical that affects mood and contributes to obsessive behaviors. But it wasn't clear whether the serotonin imbalance was caused by the eating disorder or existed before the bulimia began (Joseph, 1998).

Kaye (as cited in Joseph, 1998) measured spinal fluid, and found that the former bulimics' had abnormal levels --twice the average level --of a by-product of serotonin called 5-HIAA, which told him that there was too much serotonin in their bodies.

Bingeing and purging actually reduced serotonin activity, so Kaye (as cited in Joseph, 1998) speculates that those activities may be bulimics' unconscious way of trying to regulate serotonin levels and relieve the anxiety and obsessive behavior (Joseph, 1998). His finding is only one of several on the horizon that may persuade many more scientists to change their opinions as to the true cause of bulimia and other eating disorders. "When it comes to bulimia" says Kaye, (Joseph, 1998, on-line) "We're in the same stage where schizophrenia was 20 years ago. People used to think [that] schizophrenia was something your mother caused, but now we know it's a brain disorder" (on-line).

In the case of the serotonin research, Kaye (as cited in Joseph, 1998, on-line) theorizes that the evidence of excessive levels of serotonin in the bodies of the recovered bulimics, but not present in the bodies of the normal women, shows that the distinguishing difference between the two groups of women is high serotonin levels. When such a renowned scientist says that in his estimation the obsessive actions of the

bulimics may be heir unconscious way of trying to regulate their serotonin imbalance, the author sees this as further evidence of the fact that the genetic theory of eating disorders may be a step in the right direction.

Bulik (as cited in Joseph, 1998, on-line) has this response to Dr. Kaye's theory: "[His] research clearly shows that we can't simply blame eating disorders on the 'thin is in' ideal our society has created. Over the next five years . . . I am certain we're going to turn the corner, where the entire mental health field agrees" (on-line).

While Dr. Bulik agrees that: "genes load the gun" (Chamberlain, 1998, on-line) in chronic cases of problem eating, she insists that social and environmental circumstances do initially, "pull the trigger" (on-line). Her growing interest in this genetic theory, led Bulik et al. (1998) to conduct a study on the causes of bulimia using identical and fraternal twins, since, as this report mentioned earlier, identical twins have 100 percent identical genes [and] fraternal twins have only half in common. She found that one could deduce that if bulimia in both twins is more common among identical pairs than fraternal pairs then the reason for the disease is genetic.

In this twin study, the doctor and her colleagues interviewed 1,897 female twins at age 30 then again at age 35 and used the results to separate "genetic factors, shared environmental conditions like parenting, unique social influences (something that one twin experienced, but not the other) and socioeconomic factors" (Chamberlain, 1998, on-line). It turned out that "only 17 percent of susceptibility could be traced to a twin's unique experiences. For example, a twin who becomes a gymnast might face more pressure to be thin than her sister" (on-line) the researchers say.

Contrary to traditional belief about eating disorders, Bulik did not find that parenting was a determining factor in the results of her experiment. At the time of her twin study, Dr. Bulik did not have sufficient evidence to speculate that her theory about anorexia's genetic roots also holds true for other eating disorders, as well, but she subsequently deduced that anorexia does run in families.

Bulik's study (as mentioned in Chamberlain, 1998, on-line) with anorexic individuals, mentions one young lady in particular, Tracy (this name is assumed), "who was 20 years old before she realized how healthy, normal women eat. During a period of hospitalization for anorexia, her body dangerously drained by her refusal to eat, she watched in awe as a group of nurses sat down nearby with trays full of cafeteria food and finished every bite" (on-line).

According to Chamberlain, Tracy said, "that's just not what I grew up with. My mom has always been very weight conscious, and she's always been underweight. For lunch, she'd have black coffee. That's it. One time a friend took her to lunch for her birthday, and she had salad, with no dressing and coffee" (on-line).

Tracy has battled continually with anorexia and bulimia, according to the Chamberlain article. Yet, "she knows she's not the only one in her family with an eating disorder. That's why she wasn't surprised when Dr. Bulik told her the problem might be in her genes" (on-line). Bulik's conclusion (as cited in Chamberlain, 1998) is that genes account for 83 percent of female susceptibility to the disorder.

Although other studies show that eating disorders run in families, it was not until Bulik's study (as cited in Chamberlain, 1998, on-line) that researchers became more certain that it was genetic inheritance rather than "the same dysfunctional environment

women shared" (on-line) which caused the disturbance in their eating habits. A later chapter will relate the story of a young, gay man whose impression still is, that his mother was instrumental in his career as a binge eater because throughout his childhood she ignored the fact that he ate enormous quantities of food. This belief is also prevalent among some doctors who believe that the tradition of looking at eating disorders as a major social issue is correct.

Evidence from Kaye's serotonin study (as cited in Joseph, 1998, on-line) and Bulik's identical twin research (as cited in Chamberlain, 1998, on-line) certainly cast a new light on traditional attitudes toward eating disorders.

The following review of the findings of a "Controlled Family Study of Anorexia Nervosa and Bulimia Nervosa: Evidence of Shared Liability and Transmission of Partial Syndromes" (Strober, Freeman, Lampert, Diamond, & Kaye, 2000) will show that recently documented research says women who have a relative with an eating disorder, are more likely to suffer from the disease than their contemporaries who have no family history of either anorexia nervosa or bulimia.

In March of this year, researchers from the Department of Psychiatry and Bio-behavioral Sciences and the Neuropsychiatric Institute and Hospital at UCLA (Strober et al., 2000) joined forces to study genetics and eating disorders. The details of their research follow.

The objective of the Strober et al. study was to determine the lifetime rates of full and partial anorexia nervosa and bulimia nervosa were determined in first-degree relatives of diagnostically pure proband groups and relatives matched, never-ill comparison subjects. The researchers found that whereas anorexia nervosa was rare in

families of the comparison subjects, full and partial syndromes of anorexia nervosa, the relative risks were 11.3 and 12.3 in female relatives of both anorexic and bulimic probands, respectively. Bulimia nervosa was more common than anorexia nervosa in female relatives of comparison subjects, but it, too, aggregated in the families of ill probands; the corresponding relative risks for bulimia nervosa were 4.2.

Dr. Barbara Rost, Medical Researcher at the University of Basle in Switzerland, is also heading in the direction of genetic predisposition with her research on eating disorders. Rost (as cited in "Gene theory on eating disorders", 2000, on-line) believes there are differences in the structure of the brains of people with eating disorders.

According to this on-line article, Dr. Rost compared the brains of individuals affected by disordered eating, to people without eating disorders, by the use of a MRI scan, in order to illustrate her theory that eating disorders run in families (on-line).

Quite apart from the abovementioned studies (and others) showing altered brain chemistry and twin-related genetic predisposition, there is further evidence to throw cold water on the traditional point of view that body image disturbance is sufficient to push individuals over the edge of dieting and into the arms of death.

For example, medical practitioners are currently diagnosing very young children, who have no interest in patterning themselves after the thin ideal media image, with serious eating/feeding disorders (Raphael, 2000, on-line). "Experts estimate that as many as two in a hundred children will bite, scream or vomit to avoid eating. Such children are not just picky eaters; they are suffering from feeding disorders, a newly recognized phenomenon that specialists are just beginning to understand" (on-line).

Dr. Cathleen Piazza a School Psychologist who works primarily with children, (as cited in "My child won't eat", 2000), explains the presence of such young individuals in the controversy of disordered eating: "this is an area that has been under-researched, and it's very important that we engage in more research to understand why feeding disorders occur" (on-line).

In a more in-depth explanation of eating disorders in the very young, Piazza (as cited in Raphael, 2000) says: "Some of these children have experienced medical problems that have caused eating to be painful" (on-line). Yet, according to the article, thousands of other young children who have no history of physical problems still refuse food. "It appears that these children don't get hungry," (on-line) says Piazza. "These children are simply not motivated to eat and will go to great lengths to avoid eating" (on-line).

What makes it more imperative that answers are soon found, is: "there really are children out there, [who] if left untreated, would eat nothing by mouth and would eventually die," warns Dr. Piazza, (as cited in Raphael, 2000, on-line).

This feeding disorder specialist, who is also attached to the Marcus Institute in Atlanta, points out "I think the common notion is that children do grow out of out of bad eating habits. However, the reality is that millions of Americans suffer from health problems that are impacted by their poor diet and nutrition [from] childhood. So it probably is the case that many of these individuals were poor eaters in childhood" ("My child won't eat", 2000, on-line).

As interest in the role of genes in disordered eating mounts, researchers at The University of Pittsburgh School of Medicine, Center for Overcoming Problem Eating and Eating Disorders Clinic (COPE) are currently conducting several studies.

One of these is an international study (COPE – Eating Disorders Research, 2000) to determine whether there is, indeed, a specific gene(s) that predispose(s) individuals to develop bulimia. COPE is looking for families in which at least two members, including, sisters, brothers, aunts, uncles, cousins, or other relatives, suffer from eating disorders, to participate in discussions about their eating habits; and any related emotional problems.

Participants must complete questionnaires and provide blood samples (about 4 table-spoons in each case) for genetic analysis; and according to the clinic, there is no need for traveling, since the process can take place in the home setting. As an incentive for reluctant individuals, there is an offer of a \$150 for each participant in this study. Also, participating parents will each receive \$50, in return for observation reports and a blood sample of the family member being treated.

Another study (COPE, 2000), to determine the role of serotonin in the control of hunger is also on the COPE agenda these days. The scientists involved in the study are looking for “women between the ages of 18 and 40, who have suffered in the past from bulimia nervosa or anorexia nervosa” (on-line). As a requirement of this study, participants must complete screening and questionnaires; and agree to be hospitalized for one night. The ‘PET’ Studies (on-line) as they are called include an MRI, PET scans and blood draws. Each participant will receive \$300 for completing the assessment.

All of these plans for future research, taken with existing studies that show altered brain chemistry and familial patterns of eating-disorder patients, encourage progressive medical practitioners to look forward in anticipation, to more precise answers and new directions for treatment, in these disorders, as the new millennium unfolds. For, indeed, as Dr. Bulik (as cited in Chamberlain, 2000) puts it: "It's a real and complex biological disease, not simply desperate girls wanting to look like supermodels" (on-line).

From facts gained during research, the author believes that researchers with this progressive point of view, will most likely seek to isolate the gene(s) contributing to the disturbance and replace them with healthy genes to continue proper functioning of the chromosomal network; or in some other way, eliminate their negative effect on those centers in the brain responsible for hunger. In the author's estimation, this goal seems more possible, since the recent breakthrough in mapping the entire human genome alphabet.

With all the progress in genetic studies of disordered eating patterns, it is not surprising that an article ("Pigs offer anorexia clues", 1999, on-line) published in June of that year, brings animals into the picture: "Pigs that are bred to be slim to satisfy consumer demand for lean bacon are more sensitive to stress" (on-line). Personnel involved in this study say that lean pigs are helping them unravel the origins of anorexia because the leaner animals are showing "anorexia-like symptoms."

According to "Pigs offer anorexia clues" (1999), these researchers told the annual meeting of their colleagues in Birmingham, that four percent of the pigs exhibited classic signs of anorexia nervosa, such as over-activity and not eating" (on-line).

The study of slim pigs shows genetic traits that help them maintain slimness over and above their counterparts without the same genetic markers. As a result, British researchers believe anorexia might be inherited.

Experiment With Sisters

The article ("Pigs offer . . .", 1999) further explained that during the meeting of psychiatrists in Birmingham, Dr. Janet Treasure of the London Institute of Psychiatry presented a study of 48 pairs of sisters. One person in each pair exhibited anorexia. Therefore, Treasure (as cited in "Pigs offer . . .", 1999) a senior lecturer at the Institute says: "There does seem to be a genetic disposition to leanness in the families of anorexics" (on-line).

Treasure (as cited in "Pigs offer . . .", 1999) supports her belief with the statement that risk factors such as dieting and overweight are absent in her experiment with the sisters. "What we found was a high level of perfectionism, low self esteem and a strong need to comply", says Treasure (on-line). Treasure further explained that the sisters with anorexia were more likely to say things like "my parents always want me to achieve".

On the other hand, those without anorexia were unlikely to feel the need to comply, or would simply dismiss the expectation. So, although there were mitigating social circumstances that might have affected all of the sisters, it appears that only those individuals exhibiting the variant gene went over the edge of normal dieting practice. In the final analysis, Treasure (as cited in "Pigs offer . . .", 1999) told the Conference that in all of these studies, the focal point is the role of the genes in distributing this chemical

substance called 'serotonin' which helps to control mood. This was a further shot in the arm to the growing belief in the theory of genetic predisposition in eating disorders.

However, Treasure, (as cited in "Pigs offer . . .", 1999) like Bulik et al. (1999) was very cautious about discounting entirely, the possibility of a role played by social circumstances:

It seemed as though the sisters with anorexia were more likely to have variations in genes related to serotonin. . . [and] it's important to realize there is a mixed vulnerability to anorexia. It's not due to genes or environment, but rather a combination of the two factors . . . anorexia nervosa is a complicated disorder and genes aren't everything. The genes load the gun but the environment pulls the trigger (on-line).

Summary

To summarize the position of the gene theory of eating disorders, a study quoted in an April 14, 2000 article in BBC Health News, ("Gene theory on eating disorders", 2000) "a particular gene might make someone more vulnerable to eating disorders such as anorexia nervosa and bulimia" (on-line).

According to a report from the annual conference of the British Psychological Society, ("Gene theory on eating disorders," 2000) "a particular gene might make the brain more sensitive to chemicals [that] regulate appetite and mood" (on-line).

In fact Dr. David Collier (as cited in "Gene theory . . .", 2000) from London's Institute of Psychiatry reported that the Gene in question is "shown to be twice as common in women suffering from the disorder" (on-line).

But, if none of these current genetic findings puts the theory of the 'thin ideal' body image for women to rest in this controversy of disordered eating, information that even in societies where fat people are seen as beautiful, individuals suffer from eating disorders ("Genetic clues . . .", 1999) should send a strong message to medical practitioners who base their belief entirely on the traditional ideas of body-image disturbance.

The results of this fat-is-beautiful study came from medical researcher Dr Hans Hook (as cited in "Genetic clues . . .", 1999) "who used to believe that anorexia only existed in Western countries ... where there was pressure to be thin" (on-line). Then he went ahead and conducted a study on the Caribbean island of Curacao, where fat is considered beautiful. The results of his experiment did not support the idea that societal stereotypes are a causative factor in eating disorder issues. This author feels that the preponderance of medical research should be directed toward creative genetic intervention for individuals seriously affected by genetic disorders such as bulimia, binge eating and anorexia nervosa; and she challenges today's cutting-edge biogenetic medical community to continue to unravel the mystery of the exact role of the brain's 'master molecule' -serotonin--in eating disorders such as anorexia nervosa, bulimia and binge eating.

Chapter III

TRADITIONAL IDEAS

Introduction

This chapter discusses the fact that while there is much progress in the search for genetic links to these diseases, "there are still a lot of old fashioned ideas about eating disorders floating around," (Old Fashioned Ideas, 1998, on-line). The table below shares some common misconceptions, then destroys them, in order to dispel the old myths surrounding eating disorders.

Table 1

Common Misconceptions About Eating Disorders

Misconception	Rebuttal
Victims are young; white; female	Any-one can suffer
Sufferers are extremely thin	Most are average
Dieting is vanity	Eating disorders are vanity
Compulsive overeaters are lazy	Low self esteem is present

Note. As far as society has come in the past few years there are still a lot of old-fashioned ideas about Eating Disorders floating around (Old Fashioned Ideas, 1998)

Chapter III also uses articles and case studies to illustrate the traditional point of view on the issue of eating disorders; and analyzes them in the face of new genetic

studies. But sometimes old habits (and beliefs) die hard. As a result, some medical researchers, as well as some individuals affected with eating disorders prefer to believe that these diseases are caused primarily by body image disturbance.

New Studies/Old Images

As recently as September 1999, Dr. Claire Mysko (as cited in Bennett, 2000) Administrative Director of the American Anorexia Bulimia Association, in an article entitled: "Anything To Be Thin," refers to the fact that a woman's struggle to stay thin – eternally, is providing some industries with lucrative earnings.

"There is a lot of money to be made from women's insecurities," (on-line) the director explains on the ABC website for persons with eating disorders. Mysko (as cited in Bennett, 2000) also feels: "too many women [even slightly overweight women] believe that if [one is] thin and beautiful, [one is] automatically happy" (on-line).

The Ballet Physique

The belief that eating disorders develop mostly because of social and environmental circumstances is also stated on the Something Fishy Website on Eating Disorders (www.sfwed.org) as part of a discussion on anorexic ballet dancers:

With many pressures on a ballet dancer, the greatest pressure is being lean. This pressure is what drives a dancer to be anorexic. A ballerina has many pressures on her, but the pressure to be thin comes before all others. The pressures of media are the first pressures that a young girl

will notice when developing into a young woman. She will be looking through a catalogue looking for new clothes and sees that all of the models have beautiful little figures (on-line).

She will see pretty girls with no acne or noticeable birth- marks. She will get the impression that pictures are how people should look when they become older. As a young dancer gets older, she will see pictures of the best dancers in the world. They are characterized with narrow hips, little or no fat deposits, slim middle, small breasts, delicate-looking arms and their height is short. A young dancer who views this feels that unless she shares these characteristics, she will never be the girl in the picture. The media pressure girls to be perfect. They do not display people who are anything but the ideal, and this can have a lasting effect on young girls.

Dance teachers also pressure their young students to be like their heroines. In classes they are told to hold up their stomach, making them look thin from a side view. Once during class, Kristi, who was interviewed, was told by her dance teacher not to eat before class because it made her look fat. This put her off eating, because she went to over seven classes a week. The girls look up to their dance teacher, whom as their mentor has the control to forecast the girls' outcome of eating patterns. If she makes it an important issue . . . that to be thin is to be a good dancer, then the girls are more likely to become anorexic (on-line).

Proponents of the 'pressure-to-be-thin' reason behind eating disorders, claim that mothers definitely play a role in how young females develop anorexia nervosa, bulimia nervosa and binge eating disorders. The Something Fishy Website (1998) reports:

Pressures put on to a girl from her parents are hard to deal with. If a girl entering adolescence still has some baby fat on her, she may not realize that [her fat is necessary for] her body to fully mature into womanhood. This lack of knowledge may deter her, and [she may] think that she has to get rid of this extra fat . . . If parents tell their children that they are fat and need to lose the weight the children listen. As far as they are concerned their parents are always right and would never tell them something that [is incorrect]. Parents who allow their daughters to feel fat because of something they may have said are just as much to blame for the development of the disease as the daughters. This is because the m daughters have always looked up to their parents. If their parents [find] fault with their daughters and their figures the daughters will immediately respond by losing weight (on-line).

It is true that Strober, M., Freeman, R., Lampert, C., Diamond, J. & Kaye W., (2000) of the University of Pittsburgh, conducted a study which shows that environment does play an important role in eating disorders. However, this study does not place the blame totally, or even predominantly on environmental causes. Strober et al.'s (2000) study, first published by the American Journal of Psychiatry, reports that: "Whereas

anorexia nervosa was rare in the comparison subjects, full and partial syndromes of anorexia nervosa aggregated in female relatives of both anorexic and bulimic probands, respectively.” Strober and his team concluded that: “Both anorexia nervosa and bulimia nervosa are familial.”

Similarly, Wade, T., Bulik, C., Neale, M. & Kendler, K. (2000) conducted a study to estimate the degree of heritability for anorexia nervosa, and also to examine the origin of a co-morbid relationship between anorexia and major depression. Wade et al.’s results found that: “anorexia nervosa was estimated to have a heritability of 58% contribution of shared environment [and that] the co-morbidity between anorexia nervosa and major depression is likely due to genetic factors that influence the risk of both disorders” (Wade et al., 2000, on-line). Then Mundell, in a report published by Reuters Health Information News Service, quotes Strober as saying: “it remains unclear which factors play a more important role –family environment or genes – when it comes to ‘clustering’ of eating disorders within specific families” (Mundell, 2000, on-line). According to the Mundell article, Strober notes: “current research is searching for more specific areas of the genome that might be associated with risk to these conditions” (on-line) to ensure that the causes of eating disorders are not due to outside pressures.

Notwithstanding current research findings about the role of genetics in eating disorders, even individuals who suffer from these diseases cling to the stereotypes about social pressures, never considering familial or genetic tendencies. In a 4000-word essay (“Eating disorders in ballet dancers”, 1998) one recovered anorexic patient places the blame squarely on the dancing industry that she says places inordinate pressure on dancers to be thin.

The essayist speaks harshly of her profession, which she says finds it appropriate to reinforce the thin ideal image. The dancer points out, "a select group, young female ballet dancers are molded into thinking that the only way to succeed with their dream is to be thin" (Eating disorders . . . , 1998, on-line). She promises: "the causes of anorexia will give an indication of why ballet dancers are at high risk of getting this disease [and] it will explore what drives them to be thin and why no simple means will end their obsession" (on-line).

The survivor of anorexia ("Eating disorders in ballet dancers", 1998, on-line) writes that anorexia nervosa is a "serious psychological disorder due to cultural ideas of feminine beauty . . . [which] causes young women [to] feel a strong desire to be thinner than their bodies naturally tend to be" (on-line).

She credits her information to the Counseling Center of the State University of New York and Buffalo and the Counseling Center of the University of Virginia. The recovered victim of mild anorexia says that according to the experts, the onset of the disease is either 11 years (the onset of puberty) or 18 years (the beginning of adolescence) "Both beginnings to a new phase in life" (on-line).

This belief that eating disorders have their foundation in body image disturbances is even encouraged by dictionary definitions. The American Heritage College Dictionary (3rd. ed.) (1993), currently defines anorexia as: "a disorder, usually occurring in teenage women, characterized by a fear of obesity, a distorted self-image, an aversion to food, and a severe weight loss" (p. 55). Bulimia is defined as: "an eating disorder characterized by episodic, uncontrolled binge eating" (p. 185), with no mention of possible genetic predisposition. Hence is it not difficult to see why many members of

the medical profession and affected individuals, themselves, do not consider that these illnesses are a genetically caused problem.

For Women Only

Although females are the primary focus of traditionalists in this controversy over the precise causes of bulimia, anorexia, and binge eating, homosexual males "who are less satisfied with their bodies and choose a thinner ideal are also at risk for serious eating disorders" ("Eating disorders are not just a woman's problem", 1997, on-line). So, the proponents of this traditional point of view are at least willing to concede that eating disorders are not just a woman's problem because, "a disproportionate number of males being treated for eating disorders are gay" (on-line).

What's more, Ian Williamson (as cited in "Eating disorders are not just a woman's problem", 1997, on-line) conducted a study aimed at about 100 gay and straight men. The subjects were given a series of nine drawings of men, ranging from the very fat to the very thin, to select their ideal drawing as well as the one who looked most like them. Williamson's study (on-line) found that "the gay men were less satisfied with their bodies and chose a thinner ideal body" (on-line). The study concluded that gay men do not come forward in larger numbers to discuss their eating problems because "the idea of having a 'feminine' disease can deter any man from seeking help and gay men can be more reluctant, fearing their sexuality might be put under a microscope" (on-line).

The article quoted more precise statistics: "Researchers in America say that 90% of males treated for eating disorders are homosexual; but their study, based on a small

number, it is unlikely that the findings are representative" ("Eating disorders . . .", 1997, on-line).

Frances Young, a Manchester counselor who specializes in eating disorders among men (as cited in "Eating disorders . . .", 1997, on-line) found that about one-third of her clients are gay. But she emphasized: "for people to get the help they might need, professionals need to look out for eating disorders in both gay and straight men" (on-line).

"Men might be picked up for stress or problems at work and then doctors might think they're off their food because of the stress. It's seen very much as a white middle-class, female problem, and can be missed altogether in men," according to Young (on-line).

However, genetic studies mentioned in the preceding chapter show that genes and brain chemicals do play a very important part in the drive for hunger; and further, that gender and/or body image disturbance are simply cosmetic issues outside of the real cause for these disturbances. "The most important thing, overall, is to remember that most of the underlying psychological factors that lead to an eating disorder are the same for both men and women" ("Issues for men with eating disorders", 1998, on-line).

"Issues for men with eating disorders" (1998) also says:

Only recently has the media begun to address the 'hidden population' of men with eating disorders. Many men suffer but because of the old wives' tale that eating disorders [are] just illnesses of women, the men suffering have feared telling of their problems, or even facing it

themselves. It is calculated that 1 in 10 eating disorders currently are men, and [we believe] that the number is much higher. [The article continues]: These are just some of the fallacies put out to society by the media and the medical community. It is time to stop the ignorance. Eating disorders and the people who suffer from them are valid and not to be taken lightly. People die from this and it is not our choice to live this way throughout our lives (on-line)

Drs. Williamson and Mysko are not the only scientific researchers who still look toward body image disturbance as the primary cause of eating disorders. Drs. Madhulika A. Gupta and Andrew M. Johnson (as cited in "Dissatisfaction with . . .", 2000) of the University of Western Ontario in Canada study 53 women with anorexia and/or bulimia and 73 women without eating disorders, to see whether the groups differ in their perception about non-weight issues such as: skin, teeth, jaw-line, nose, eyes, ears, hair, and height. The women (all under 40 years) answer questions related to their level of satisfaction with the features mentioned; and completed an eating disorders questionnaire. The eating disorder respondents showed "high levels of body dissatisfaction and preoccupation with thinness" (on-line).

Gupta & Johnson, (as cited in "Dissatisfaction with . . .", 2000) further suggests, that individuals with these disorders also tend to be unhappy with other features of their bodies that have nothing to do with weight (on-line). These findings appeared first in the: International Journal of Eating Disorders, and the researchers did mention that "experts have been unsure whether women with eating disorders who also express

distorted views of other body parts have an additional illness that needs to be treated" (on-line) or whether these symptoms are part of the overall eating disorder. However, no mention was given to the possibility of genetic causes. In fact, Gupta and Johnson (as cited in "Dissatisfaction with . . .", 2000) believe "dissatisfaction with non-weight parts of the body is part of anorexia and bulimia - and says this information "fits in with previous research showing that the best predictor of successful treatment in eating disorders is not actually eating behavior, but the degree of body image disturbance" (on-line).

Journeys in Bulimia

The traditional trend of attributing eating disorders simply to body image disturbance is supported by a mother-daughter journal printed in October 1998 because it reflects the traditional point of view shared both by some members of the medical profession, as well as victims of eating disorders and their families.

Rebecca Cohen and her daughter Toby Levin (mentioned in Chapter II), published: *A Mother –Daughter Journal* (Cohen, & Levy, 1998), which discusses the difficulty, faced by victims; as well as family members who must nurture a loved one through an eating disorder. (The reader is referred to Cohen and Levy (1998) for a narrative of experiences typical of the struggle of individuals with eating disorders.)

Summary

While this author empathizes with the pain and frustration of Tobin and her family, she feels that the greater tragedy would be for Ms. Levy to continue her struggle unaware of the trend toward genetic research and the lasting implication that treatment designed along genetic lines could have for her condition.

Chapter IV

CONCLUSIONS AND FUTURE RESEARCH

This chapter begins with a detailed account of the action of serotonin on human behavior then reviews research abstracts on the subject of genetic causes of eating disorders and current treatment options with a view to determining the effectiveness of the genetic approach. The author uses tables to show the successful use of serotonin re-uptake inhibitors that are designed to remove excessive levels of serotonin from the body, and return them to the brain where they can prevent obsessive behaviors like problem eating and other compulsive disorders.

In this final chapter the author introduces the idea of a possible co-morbidity between disordered eating and major depression, since she believes the latter illness happens before the onset of eating disorders such as anorexia nervosa, binge eating and bulimia nervosa even begin to make an appearance in the lives of affected individuals. This author tried to ensure that all statements concerning co-morbidity between these two diseases are supported by documented research findings.

The Serotonin Effect

According to 5- HTTP: The natural way to overcome depression, obesity, and insomnia (Murray, 1998):

Scientists first discovered and isolated serotonin from the blood in the 1940's. Research since then confirms that serotonin . . . is found in

platelets, the component of blood that promotes clotting. Whenever you bleed, the platelets release serotonin to help constrict the blood vessels and minimize further loss of blood. The cells of your digestive tract also contain serotonin and serotonin receptors. This chemical regulates the secretion of stomach acid and other digestive fluids. It also stimulates certain intestinal muscles to contract, which helps move food along.” (p.16).

However, Murray points out that:

Our main concern . . . is with the role of serotonin within the brain. Serotonin influences a wide range of normal brain activity, including moods, eating patterns, pain transmission, sexual behavior, and sleep. The level of serotonin present in one's brain can have a tremendous impact on how one thinks feels and behaves. Having an adequate supply produces what is sometimes called the 'serotonin effect' –a feeling of calmness, mild euphoria, and relaxation . . . Too little serotonin can lead to the opposite situation –feelings of depression, anxiety, and other problems associated with serotonin deficiency syndrome (p. 44).

In addition, the author draws attention to the fact that most of the problems associated with serotonin deficiency are common to both major depression and eating disorders, hence the belief that there is some kind of relationship between the two

diseases. Studying possible co-morbidity between these illnesses, however, becomes more challenging when one learns that there are: (a) different types of serotonin receptors in the brain and; (b) that each individual has a unique reaction to the same serotonin deficiency according to individual biological differences (Murray, 1998). The author also reveals that scientists recently found at least seven (maybe even 15) subtypes of serotonin Receptors embedded in the brain; and explains that receptor concentrates on a different function in the body. For example, one kind of serotonin receptor handles our response to anxiety, while another is in charge of blood vessel constriction. The lower one's level of serotonin, the more severe and widespread, the potential impact on the brain and body. For example, low levels of serotonin can cause overwhelming sugar cravings. Research has also shown that many people with bulimia, an eating disorder that causes uncontrollable eating binge have insufficient levels of serotonin. Low serotonin levels also are involved in depression—a common and often serious mental disorder characterized by very low mood and reduced levels of functioning.

Some scientific studies have found that people with the lowest levels of serotonin are at greatest risk of attempting or committing suicide. One of the difficulties associated with treatment of this deficiency is that there are variations in the response of different individuals to the same level of serotonin. In some people, low levels of serotonin may cause depression, while in some others the same level might produce regular disabling headaches or a voracious appetite for sweets and carbohydrates (Murray, 1998, p. 19).

Notwithstanding these major differences in how individuals respond to signals sent to the electrochemical system of the brain, “the bottom line is that if we hope to live

a healthy happy life, we need proper and balanced levels of key brain chemicals”
(Murray, 1998, p. 19).

It is important to realize that in the use of serotonin, medical practitioners cannot simply inject necessary quantities of it into the systems of their subjects, because this neurotransmitter has to be manufactured inside the brain from other raw materials, in order to be effective. “And the best source of those raw materials is 5—HTP . . . one of the most exciting breakthroughs of this or any century” (Murray, 1998, p. 20). Murray, 1998, seems quite certain that by regulating the production and distribution of serotonin in the brain, he can virtually relegate such diseases as eating disorders and major depression to the medical and scientific archives, thereby saving millions of women from certain death.

Eating Disorders –The Bare Facts

Initial warnings against the long-term effects of old, psychosocial treatment options involving therapy, counseling sessions and psycho-social relationships began to surface about a year ago, when research findings by prominent medical researchers were published. The medical researchers pointed out that although some progress has been made in the understanding and treatment of [eating disorders] “a substantial proportion of individuals who suffer with these illnesses, have a limited response to treatment.” (Kaye, Strober, Stein & Gendall, 1999) According to the findings in the research Kaye et al. (on-line) “treatment strategies used in eating disorders have tended to be adopted from therapies that were devised to treat other psychiatric illnesses” (on-line). Ongoing studies “suggest that eating disorders are independently transmitted familial liabilities

with a unique pathophysiology” (Kaye et al., 1999, on-line). This means that the researchers believe medical doctors who approach the treatment of eating disorders as if the individual had become a disordered eater because of societal pressures on women to be thin; or because a male is gay, and also affected by body-image disturbance; or perhaps because families foster poor eating habits, are likely to achieve limited success. An independently transmitted familial liability suggests that within each family’s unique genetic makeup, some malfunctioning gene or genes can cause an individual family member’s genetic cycle to falter, thus creating inappropriate emotional responses like mood swings or eating disorders.

According to Kaye et al. (1999): “These new findings raise the possibility that an improved understanding of the pathogenesis of eating disorders will generate more specific and effective psychotherapies and pharmacologic interventions” (on-line). In other words, once doctors gain greater insight into the genome system, they will better be able to design more lasting solutions for the treatment of eating disorders

Prominent medical research institutions such as: the Western Psychiatric Institute, the University of Pittsburgh School of Medicine and Department of Psychiatry and Bio-behavioral Sciences and the Neuro-psychiatric Institute and Hospital at UCLA, are a few of the American institutions currently challenging the old fashioned ideas about eating disorders as they feverishly seek cures for these troubling disorders.

Several family and twin studies, including research done by Strober et al. (2000), Kaye et al. (1999), Bulik et al. (1999), Drs. Hans Hook and David Collier (as cited in “Genetic clues . . .”, 1999) and Dr. Janet Treasure (as cited in “Pigs offer . . .”, 1999) are steering traditional research away from ideas of feminine ideal body images

portrayed in the media, to educate the global medical profession about the potential role of genetic transmission in anorexia nervosa, bulimia and binge eating.

Because of the revealing results of current medical research, this author believes that the recent mapping of the entire human genome system, will hurry geneticists on to the answers they so earnestly seek and might even provide further information about the relationship between disordered eating patterns and major depression.

In support of the idea of co-morbidity, a research abstract published in the American Journal of Medicine, (Wade, Bulik, Neale, & Kendler, 2000) concluded that although the study in question “was limited by the small number of twins, the results suggest that genetic factors significantly influence the risk of anorexia nervosa, and substantially contribute to the observed co-morbidity between anorexia nervosa and major depression” (on-line). These researchers at the Virginia Institute for Psychiatric and Behavioral Genetics, at Virginia Commonwealth University, “sought to derive heritable estimates for anorexia nervosa and to explore the etiology (the cause or origin) of the co- morbid relationship (a second condition existing along with an initial diagnosis and seeming to affect each other) between anorexia nervosa and major depression” (on-line). The researchers “applied bivariate structural equation modeling (compared the genetic make-up of an individual that has two related diseases) to a broad definition of anorexia nervosa and lifetime major depression, as assessed in a population based sample of over 2,000 female twins” (on-line). They found that: (a) Anorexia nervosa was estimated to have a heritability of 58% contribution shared environment and (b): The co-morbidity between anorexia nervosa and a major depression is likely due to genetic factors that influence the risk of both disorders.

When one considers a study by Bulik, Sullivan and Joyce (1999), who measured temperament, character, and suicide attempts in anorexia nervosa, bulimia nervosa, and major depression the co-morbid relationship between the eating disorders and depression becomes even clearer. The researchers “compared the prevalence and severity of suicide attempts of women with anorexia nervosa, bulimia nervosa and major depression, with no history of an eating disorder, and . . . examined the TCI scales and suicide attempts. They found that comparable women across the three groups had attempted suicide” (on-line). The researchers concluded that suicide attempts are equally common in women with eating disorders and women with depression.

In the author’s estimation, the idea of co-morbidity is also evident in an account of factors specific to, factors contributing to, and treatment associated with, eating disorders. Powell, (1996) listed depression and anxiety as common factors; genetic predisposition and biochemical causes; and psychotherapy and antidepressant medication as possible treatment for anorexia and bulimia.

Similarly, Michael Murray, a doctor of Natural Medicine, lists anxiety as a common symptom in various kinds of depression and The American Medical Association Essential Guide to Depression (AMA, 1998) points out:

For many years, doctors have known that depressive illness runs in families. Many people with major depression or bipolar disorder can name family members who also struggle with the illness. However, for a long time, there was no way to know for sure if people inherited a susceptibility to depressive illness or if the illness was caused by

something in the environment. Today, researchers understand that depressive illness is to some extent inheritable . . . a unit of heredity called the gene carries this tendency. (p. 76).

The similarities between eating disorders and major depression continue with information that 5-HTP (also used to treat eating disorders, successfully) is a successful treatment for individuals suffering from depression: "researchers confirmed their theory that antidepressant therapy with 5-HTP is much more effective in patients who have low levels of 5-HIAA (a chemical that is left over when the body breaks down molecules of serotonin) (Murray, 1998, p. 60).

Before this chapter concludes, the author finds it appropriate to discuss in greater detail, how serotonin, the major factor in both depressive illnesses and eating disorders, works to keep the body healthy. Table 2 shows that both mental outlook and eating patterns are affected by variations in one's level of serotonin. (Murray, p. 17).

Table 2

Effects of Different Levels of Serotonin

Optimal level of serotonin	Low level of serotonin
Hopeful, optimistic	Depressed
Calm	Anxious
Good-natured	Irritable
Patient	Impatient

Reflective and thoughtful	Impulsive
Loving and caring	Abusive
Creative, focused	Blocked, scattered
Able to think things through	Flies off the handle
Responsive	Reactive
Does not overeat carbohydrates	Craves sweets/high carbohydrates

Note. "If something goes wrong with the serotonin system . . . the results can include plunging moods, health threatening food cravings . . . skull-cracking headaches . . . and just plain exhaustion" (Murray, 1998, p. 17).

In Support of Co-morbidity

In the author's estimation, the following conditions said to be associated with low levels of serotonin gives further credence to the theory of co-morbidity between major depression and eating disorders, because these symptoms are common to both these diseases. Table 3 lists the common symptoms.

Table 3

Symptoms Associated With Low Serotonin Levels

1. Aggression
2. Alcoholism
3. Anxiety
4. Attention Deficit Disorder

5. Bulimia
 6. Carbohydrate cravings
 7. Chronic pain disorder (such as fibromyalgia)
 8. Depression
 9. Epilepsy
 10. Headaches (migraines, tension headaches, chronic headaches)
 11. Premenstrual syndrome
 12. Schizophrenia
 13. Seasonal affective disorder ('winter depression')
 14. Suicidal thoughts and behavior
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Serotonin, of all the neurotransmitters in our body, "is, the one most involved in the onset –and for that matter, the treatment—of various medical and psychiatric problems" (Murray, 1998, p. 17). Indeed, Murray goes even further, to say, emphatically, that he: "consider[s] serotonin deficiency syndrome to be one of the most widespread and debilitating problems of our time" (Murray, p. 17). This latter statement (among others) persuades the author of this report that the magnitude of the challenge in finding effective cures for both depressive illnesses and eating disorders is great; and medical science must find immediate answers.

Evidence of Genetic Transmission

When 5-HTP is used to treat obesity, individuals consume substantially fewer calories, carbohydrates and proteins than when 5-HTP is not administered (see Table 3).

Now we must remember that serotonin is one of the busiest chemicals during normal brain activity. As Michael Murray (1998) explains, this “master molecule” plays the part of a traffic cop that co-ordinates the activity of many other neurotransmitters and also controls both mood and behavior. Murray’s book even sub-titles his section on the chemical: Serotonin: The Brain’s Master Molecule. The doctor of natural medicine then describes what happens when serotonin fails to reach desired levels, as the show-stopping occurrence of a leading actor forgetting his lines; or failing to enter on cue.

In this book about the natural way to overcome depression, obesity and insomnia, the reader learns that scientists first discovered and isolated serotonin from the brain, during the 1940’s. Further research confirmed that serotonin is found in (a) platelets, for constricting blood vessels so blood-clotting can occur; (b) in digestive tract cells where it enhances the secretion of stomach acids fluids; (c) in digestive muscles where it stimulates contractions that pushes food forward (Murray, 1998, p. 16). Whenever serotonin levels are low, all these vital processes are interrupted in their smooth functioning. This is why Murray considers serotonin deficiency syndrome . . . “the most debilitating problem of our time” (p. 142). Murray also considers low serotonin levels the most widespread problem of our time, because in 1996 over 28 million people in this country – 1 in every 10 –were taking a prescription antidepressant medication. (Murray, 1998, p. 42). More recently, a study spanning 21 years (as cited in (“Social/psychological symptoms . . . ”, 1998, on-line), conducted to measure the influence of “social, psychological, and medical factors on [anorexia nervosa]”; describes chronic eating disorder as a “psychiatric disorder of disturbed eating behavior, characterized by extreme weight loss and fear of obesity; and a dangerous illness that

can lead to death” This on-line, health article says “in Western countries, up to 15 percent of college-aged women suffer from this eating disorder which can lead to “a wasting-away of the heart muscles among other fatal diseases. But, by far the most damaging aspects of the disorder could involve the patient’s mind” (on-line).

“Social/psychological symptoms . . .” (1998) also states that some researchers believe “the disorder is a form of depression; while others attribute its development to problems in emotional development or in family dysfunction . . . but a full recovery requires a healing of the mind” (on-line). Many investigations of the role of the patient’s mind in the onset of diseases like depression and eating disorders, are beginning to focus on the fact that “we need adequate levels of serotonin to function at our best –to stay active, alert, well rested, and happy [while] low levels of serotonin can lead to depression, sleep problems, eating disorders, pain syndromes” (Murray, 1998, p. 39).

This seemingly co-morbid relationship between eating disorders and depression brings to mind the question of the chicken and the egg. Which came first? Was it the chicken or was it the egg? In the author’s estimation, optimum treatment approaches in the cure of eating disorders could depend on the determination of whether eating disorders (the egg), or depression (the chicken) develops first. By the end of this manuscript, readers may or may not come to the agreement that this question is important for further research into effective cures for anorexia nervosa, bulimia and binge eating. My hope, however, is that some scientific investigation does continue to examine the troubling (not so trivial) questions of whether eating disorder individuals subsequently become depressed because of their illness; or whether depressed individuals develop a sort of death-wish, and binge, purge, or starve themselves until

they waste away to nothing. As far as this author is aware, the global scientific community has not yet published answers to this critical question. But while the correct answer may significantly improve chances of treating these diseases effectively, the thesis topic of this report is: Are eating disorders, anorexia nervosa, bulimia and binge eating caused by genes?

Stated briefly, the term neurotransmitter refers to chemicals that transport impulses to specific cells, both inside the brain and in other parts of the body.

Secondly, serotonin—the most important neurotransmitter in the brain -- governs the activities of the appetite center, the pleasure center and numerous other critical centers of activity in the brain. Serotonin, acts as the catalyst or “traffic cop” for safe and effective manufacture of serotonin for vital body functions (Murray, 1998 p. 113).

Table 4 shows that in weight loss studies using serotonin, over a 5-week period there were substantial reductions in subjects’ use of calories, proteins and carbohydrates, but not in those subjects using a placebo.

Table 4

5-htp and Obesity Study #1: Effect Of 5-HTP and Placebo on Food Intake

	Food Intake (Calories/day)	Protein Intake (G/day)	Carbohydrate Intake (G/day)
Before treatment	2,903	101	274
Placebo	2,327	85	223
5-htp	1,819	79	176

Note. Table above shows that women in this study reduced their caloric intake by more than 1,100 calories each day, while taking 5-HTP, and lost more than 3 pounds during the 5-day period (Murray, 1998, p. 114).

Murray (1998) puts these effects of weight loss in better perspective when he explains that a subject who reduces her calories by this extent "would burn off about one and a half pounds of fat each week, six pounds in a month, and "a whopping seventy-three pounds in a year" (p 114).

This evidence of 5-HTP contribution to weight loss, coupled with findings that: (a) "There does seem to be a genetic disposition to leanness in the families of anorexics", and (b) "Researchers are particularly interested in the role played by genes involved in the body's control of serotonin, a chemical that helps govern mood", Pigs offer anorexia clues (1999), this author believes that scientists will eventually prove, eating disorders are caused by genes.

Once again, in support of the subsidiary question in this report, the author wishes to draw attention to the fact that 5-HTP is as effective in treating various sub-types of depression as it is in treating eating disorders (Murray, 1998, p. 62).

For example, in 1972, Herman Van Praag and his colleagues (as cited in Murray, 1998, p. 62) at the Psychiatric University Clinic, Groningen, Netherlands, were the first to study 5-HTP in a double blind format (neither the researchers or the subjects knew who was getting the active product) to assess the degree of improvement on depressed subjects:

The process began with a small preliminary study that involved ten [subjects] hospitalized for severe, unremitting depression that was not responding to other forms of treatment. Half of the subjects received 5-HTP at doses ranging from 200 to 3,000 mg daily, while the other half received a placebo. The results were encouraging. Three out of the five people taking 5-HTP experienced significant improvement within two weeks, while all of the patients in the placebo group got worse during the three-week study. Of particular importance was the fact that people with such severe depression –remember, they were hospitalized –usually do not experience such a quick recovery (pp. 58 -59).

In another recent study entitled: "Anorexia nervosa and major depression: shared genetic and environmental risk factors" (Wade, T. D., Bulik, C. M., Neale, M., Kendle, K. S., 2000, on-line) researchers studied "a broad range of anorexia nervosa and lifetime major depression subjects in a population-based sample of over 2000 female twins.

These scientists theorize that: "co-morbidity between anorexia nervosa and major depression is likely due to genetic factors that influence the risk of both disorders."

(Wade et al., 2000, on-line).

Summary

Studies to determine the effects of 5-HTP on obesity (Table 4) show results from treatment of eating disorders as well as major depression, with 5HTP, the chemical that manufactures serotonin in the brain.

From the degree of success encountered when treating both these conditions with 5-HTP it seems that they are related or 'co-morbid' (Murray, 1998, p. 20).

Although it would be excellent to establish that this theory of co-morbidity is true, the more important task of this report on the genetic aspects of eating disorders is to show overwhelming evidence that eating disorders are caused by genetic malfunction.

This report explains in Chapter I, that some psychologists, such as Claire Mysko, (as cited in Bennett, 2000, on-line) stated that from their observations, a phenomenon called 'body-image disturbance' leads women to become obsessed with the ideal "model-thin look" and starve themselves, or binge and purge to extremes of death – rather than accept themselves, and their own particular body type. But scientists such Cynthia Bulik, Director of the Virginia Commonwealth University (as cited in Joseph, 1998, on-line) began to re-directed their focus after Kay (as cited in Joseph, 1998, on-line) speculated that [bingeing and purging] "may be bulimic's unconscious way of trying to regulate serotonin levels and relieve the anxiety and obsessive [behavior]" (on-line). Bulik (as cited in Joseph, 1998, on-line) says: "Over the next five years, I am

certain we're going to turn the corner, where the entire mental health field agrees [that] the ultimate key to eating disorders is a combination of biology and the environment" (on-line). Consequently, this author feels that theories about the 'ideal body image' which many women pursue; and 'poor family dynamics' that lead to disordered eating habits, may become a life-threatening illness – if the genetic predisposition already exists. Her understanding is that if existing genetic makeup does not permit the re-uptake of serotonin into the brain in sufficient quantities for the body to carry out normal functions like appetite control and; emotionally well- balanced, socially-acceptable behaviors, the individual's life could end in death.

The Center for Overcoming Problem Eating (COPE 2000) is currently conducting studies on individuals who suffer from depression, anxiety, and obsessive and compulsive behaviors. The COPE studies are studying ways in which serotonin may be related to eating disorders.

A recent report from the World Health Organization (1998) tells us that developed countries like the United States of America face a more acute problem, than the developing world in the frequency of occurrence of these diseases. "Eating disorders such as anorexia nervosa, bulimia and overeating are more common in the developed countries . . . [And] obesity is increasing, especially in the younger age-group . . . over-consumption of fats and sugars are taking their toll" (WHO, 1998, p. 82). But the United States is not alone. These very issues currently challenge the rest of the global scientific community. In a sense, the struggle continues, as researchers jostle each other to present their latest theories on eating disorders. It's absolutely essential that immediate answers be found if further death among the fasting (or bingeing) females is

to be stopped. Research findings, diaries, and news articles quoted in the preceding pages, point clearly to the fact whether it is a ballet dancer or an entertainer "wanna-be"; a male wanting to attract gay lovers; or teenage girls looking for approval from family and friends, the link that separates living dieters from those who starve or binge themselves to death is that a damaged gene fails to perform it's function on cue, somewhat like a leading actor forgetting his lines, or failing to respond on cue (Murray, 1998, p. 16). In other words, the gene that sets limits for normal, healthy eating habits malfunctions, and fails to regulate the production, dissemination and re-up take of serotonin by the brain.

As an increasing number of respected scientific communities and talented geneticists continue to reveal the mysteries of the newly mapped genome system it seems absolutely certain that correct answers to the challenge of rising fatality figures among females with eating disorders and, major depression will be forthcoming, soon. And if in the search for the root cause of eating disorders (genetic or otherwise) mankind will soon be able to enjoy a better quality of life more closely approximating the ideal intended by God.

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